Trimetazidine dihydrochloride

Cat. No.: HY-B0968

CAS No.: 13171-25-0

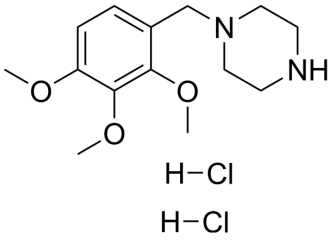
Molecular Formula: $C_{14}H_{24}Cl_2N_2O_3$

Molecular Weight: 339.26

Target: Autophagy

Pathway: Autophagy

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (294.76 mM)
DMSO : 25 mg/mL (73.69 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9476 mL	14.7380 mL	29.4759 mL
5 mM	0.5895 mL	2.9476 mL	5.8952 mL
10 mM	0.2948 mL	1.4738 mL	2.9476 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (294.76 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Trimetazidine dihydrochloride is a selective long chain 3-ketoyl coenzyme A thiolase inhibitor with an IC_{50} of 75 nM, which can inhibit β-oxidation of free fatty acid (FFA). Trimetazidine dihydrochloride is an effective antianginal agent and a cytoprotective agent, has anti-oxidant, anti-inflammatory, antinociceptive and gastroprotective properties. Trimetazidine dihydrochloride triggers autophagy. Trimetazidine dihydrochloride is also a 3-hydroxyacyl-CoA dehydrogenase (HADHA) inhibitor[1][2][3][4].

IC_{50} & Target

IC50: 75 nM (long chain 3-ketoyl coenzyme A thiolase)[2]

	β-oxidation[2] Autophagy[3] 3-hydroxyacyl-CoA dehydrogenase (HADHA)[4]
In Vitro	Trimetazidine (1 μM-100 μM; 24 hours; HUVECs) could enhance the viability of the injured HUVECs induces by oxidation in a certain dose-dependent manner[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Viability Assay[1]

Cell Line:	Human umbilical vein endothelial cells (HUVECs)
Concentration:	1 μM, 10 μM, 100 μM
Incubation Time:	24 hours
Result:	Enhanced the viability of the injured HUVECs induced by oxidation.

In Vivo

Trimetazidine (5-20 mg/kg; oral administration; 1 hour; Swiss albino male mice) in 10 mg/kg and 20 mg/kg doses significantly raises the seizure-threshold current in the increasing current electroshock seizure (ICES) test in the mice[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Swiss albino male mice (24-35 g)[4]
Dosage:	5 mg/kg, 10 mg/kg and 20 mg/kg; 10 mL/kg body weight
Administration:	Oral administration; 1 hour
Result:	In 10 mg/kg and 20 mg/kg doses significantly raised the seizure-threshold current in the ICES test.

CUSTOMER VALIDATION

- Nat Metab. 2025 Jan 2.
- Mol Cell. 2020 Oct 1;80(1):43-58.e7.
- J Adv Res. 2025 Mar 17:S2090-1232(25)00186-9.
- Cell Rep. 2024 Aug 7;43(8):114591.
- Acta Pharmacol Sin. 2022 Feb 25.

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REFERENCES

[1]. Shenghu He, et al. Protective effects of trimetazidine against vascular endothelial cell injury induced by oxidation. Journal of Geriatric Cardiology, December 2008 , Vol 5 No 4.

[2]. Jain S, et al. Trimetazidine exerts protection against increasing current electroshock seizure test in mice. Seizure. 2010 Jun;19(5):300-2.

[3]. Kantor PF, et al. The antianginal drug trimetazidine shifts cardiac energy metabolism from fatty acid oxidation to glucose oxidation by inhibiting mitochondrial long-chain 3-ketoacyl coenzyme A thiolase. Circ Res. 2000 Mar 17;86(5):580-8.

[4]. Chrusciel P, et al. Defining the role of trimetazidine in the treatment of cardiovascular disorders: some insights on its role in heart failure and peripheral artery disease. Drugs. 2014 Jun;74(9):971-80.

[5]. Hossain F, et al.Inhibition of Fatty Acid Oxidation Modulates Immunosuppressive Functions of Myeloid-Derived Suppressor Cells and Enhances Cancer Therapies. Cancer Immunol Res. 2015 Nov;3(11):1236-47.

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